VOL. 35. FEBRUARY, 1908. No. 2.

The Journal
OF
Nervous and Mental Disease

Original Articles

THE FUNCTIONS OF THE CORPORA STRIATA, WITH A SUGGESTION AS TO A CLINICAL METHOD OF STUDYING THEM.[1]

By Charles L. Dana, M.D.,
OF NEW YORK.

Anatomical and Physiological.—The corpus striatum consists of three parts, the caudate nucleus, the lenticular nucleus and the nucleus amygdalae into which the tail of the caudate nucleus runs. The caudate and lenticular nuclei are in relation to and form part of the cerebral cortex just at the anterior perforated space. The nucleus amygdalae lies in and under the cortex of the uncus. Thus the striatum is part of the cerebral cortex, though connected with parts that have no very important function.

The connections of the corpus striatum are not altogether satisfactorily known. Its most important fiber-system is the strio-thalamic. This consists of fibers that pass from the caudate and lenticular nuclei (putamen) to the thalamus, sub-thalamic ganglia and substantia nigra (Edinger). These fibers are efferent. They are in close relation with the lenticular loop.

There is a band of fronto-striate fibers connecting the frontal cortex with the striate body particularly (Marinesco) (reticulated cortico-frontal bundles of Obersteiner). These are probably fibers of association. There are also fibers which originate in the corpora striata, and which pass down into the anterior portion of the internal capsule and cooperate with the motor tracts (Wernicke). There are fibers from the cortex that pass through the corpus striatum, descending in course, and fibers from the lenticular loop pass from the thalamus up through the striatum.

---

[1] Read at the thirty-third annual meeting of the American Neurological Association, May 7, 8 and 9, 1907.

Thus, in brief, the caudate nucleus is connected by association paths with the frontal cortex and the anterior portion of the internal capsule. The caudate and lenticular nuclei are connected with each other by association fibers, and both portions of the corpus striatum by a tract which passes to the optic thalamus and to the ganglia lying beneath it, as far down as the substantia nigra.

The corpus striatum is an early development from the wall of the anterior vesicle, and its cells are more of the associative and sensory type than motor. It is most closely linked to the optic thalamus and the subthalamic ganglia. It is a ganglion which is present in the lowest of the vertebrate orders, and seems to have functional importance in fishes and reptiles when the pallium is nothing but a layer of epithelial cells (Edinger). We may suppose it, therefore, to be an older portion of the hemispheres than the cerebral cortex of man. As the cerebral cortex grew in importance, in higher vertebrates, the corpus striatum became less important relatively, and while it, no doubt, has some functions in man, they are probably of an associative and automatic character, rather than independent and specialized.

It does not seem to have been proved that the corpus striatum has any motor function except that possibly it may take some part in peripheral speech and articulation and perhaps in more general automatic movements. There is no evidence that it contains thermic centers in man, but there is evidence as will be seen later that it is connected with vaso-motor control. Double lesions are thought also to cause loss of control of the bladder. The statement that double lesions produce bulbar palsy is confirmed by some autopsies but, on the whole, has not been satisfactorily established. Edinger thinks that it may have something to do with spontaneity of movements. There are recorded spasms of forced laughter and crying in lesions of these parts, but such phenomena occur from lesions in other parts of the brain also.

Some of the more recent studies of the functions of these ganglia are recited below.

Experimental and Pathological.—Sellier and Verger (1) made experiments on dogs by electrical stimulation, and found that lesions of the caudate nucleus caused a hemiparesis of the opposite side, with sensory disturbances analogous to those caused by lesions of the cortex. There were transitory disturbances of tactile and muscular, but not of pain sense.

A. Schüller (2), experimenting on dogs, found that lesions of the caudate nucleus caused apparently a hemiplegia, but he attributed it to a lesion of the capsule.

Tounel and Raviart (3) report the case of a man, aged 45, who had a right flaccid hemiplegia, with dysarthria and dysphagia, and some psychic troubles. This had lasted for six years. They found a softening of the inner segment of the lenticular nucleus.

Stida (4), in the experimental study and review of the functions of the caudate nucleus, concludes that lesions there produce no optic symptoms.

Chailchevitch (5), experimenting on dogs with electrical currents, produced tonic tension of the muscles of the trunk and extremities of the opposite side, with some augmentation of muscular activity on the same side. Respiration was slowed and blood pressure raised.

Lo Monaco and Bellanova (6) experimented on 32 dogs, removing the caudate nucleus. Four of them lived and had hemiplegia, a slight degree of hemianesthesia, and loss of muscular sense.

In another series of experiments made two years later, they state that lesions of the caudate and lenticular nuclei cause a disequilibration of the motor centers.

Mingazzini (7), as the result of pathological observations, comes to the conclusion that the lesions of the lenticular nucleus cause hemiplegia, often very slight paresthesia, vertigo, pains in the limbs and loss of memory. These symptoms gradually improve and may disappear entirely. He also observed sometimes monoplegias, facial and facio-brachial. He thinks that the motor functions of the lenticular nucleus are only supplementary. If the lesions occur in the left lenticular nucleus, they may produce dysarthria, especially if the middle part is affected. The sensory disturbances are slight and rare. Rectal and bladder troubles slight and variable. Atrophy of paralyzed parts may occur. A pseudo-bulbar palsy may occur.

In a later communication, the same author finds that small lesions of the lenticular nucleus cause no symptoms. Lesions often cause a flaccid paresis of the opposite side; when on the left side, dysarthric symptoms, when on both sides, pseudo-palsy. It is doubtful if lesions here have any influence upon movements of the eyes, reflexes, bladder or rectum.

Löwy (8) reports a case of softening of both corpora striata (caudate nucleus and outer limb of the lenticular nucleus). This caused a general rigidity, like that of paralysis agitans, without tremor.

Touche (9) reports the case of a man, 66 years of age, who suffered from an incomplete right hemiplegia, with paresis of the lower facial and of the tongue. There was a hemorrhage on the left lenticular nucleus.

Otto Marburg (10) reports the case of a double lesion (tumor), of the caudate nucleus, which caused a disturbance of the bladder function. He finds that double lesions of the putamen may also cause paralysis of the bladder.

E. v. Czhlarz (11) reports the case of a tumor of both caudate nuclei and putamen, with bladder disturbances. He thinks that there are three centers in the brain for the bladder:

One in the cortex for voluntary movements; one in the corpora striata for automatic movements, and one in the thalami for bladder disturbances connected with emotion. Lesions must be double to cause these disturbances.

V. Bechterew (12) reports the case of a man, 38 years old, who had a sudden paralysis of the right upper lid, turning of tongue to left, disturbance of speech and tonic contractions of the left upper and lower extremities. With this he had false sensations as to the position and movement of his left arm. Lesion in the striatum.

Franceschi (13) reports a case of double lesion of the lenticular nucleus, with some involvement of the adjacent parts, but not of the O. T. or posterior segment of the internal capsule. The patient had forced crying and laughing and dysarthria, symptoms of bulbar palsy, with double hemiplegia. He thinks that the lenticular nucleus has supplementary motor functions, and that through it run thalamo-cortical paths concerned in emotive expression.

Giuseppe Pagano (14) destroyed the caudate nucleus of dogs and got reactions of anger, fear and also sexual excitement. He thinks the caudate nucleus has to do with emotional activity.

Piazza reports case of double hemiplegia with (slight) dysarthria in a senile dement aged 74. Double softening of the putamen. He collects 22 cases, and concludes that lesions of the putamen, in its middle part, cause paresis of the limbs. If the lesion is in the left side there is some dysarthria. Lesions outside the median part may cause choreic and athetoid movements.

Clinical and Pathological.—I have the notes of four cases of hemorrhage or softening of the corpus striatum which are placed on record mainly because of the rather negative evidence of localization which they furnish. One might infer only that lesions of this region produce a profounder effect upon the strength and vitality of the patient than would be expected from such comparatively slight lesions, and that at times there are disorders of the motility, such as rigidity and spasm, which are rather greater than would be expected from the character and size of the lesion. In one patient gangrene of the lungs developed, a fact of some significance in connection with the gas-poisoning case. One patient had a decided dysarthria in connection with a lesion of the left lenticular nucleus. One patient had two successive lesions of the lenticular nucleus, but continued to do his work as a laborer. The cases show that the motor symptoms (paralyses and rigidity) in these lesions are probably due to pressure. The slight speech disturbance in one case might be due to other causes than simply the striate lesion. In two of the cases the lesion was double but there was no pseudo-bulbar palsy.

Case I. Female aged 70, temporary right hemiplegia, with dysarthria lasting during illness but improving. Death on thirty-seventh day. Lesion of left lenticular nucleus. No abnormal temperature changes.

Examination on Admission.—Patient was admitted on November 15, in a stupid condition. Did not answer questions. Incomplete hemiplegia of right side. Muscles of right side rigid. Biceps contracted. Face drawn towards the left side. Tongue when protruded, deviated to the right. Sensation diminished all over same side. Reflexes active. Respiration slightly stertorous. Five days later the patient had slowly and steadily improved. Paralysis less marked. Sensation improved slightly. Breathing was markedly stertorous at times. She answered some questions, but the dysarthria rendered the answers very indistinct. She swallowed and took food. Bed-sore discovered over the sacrum. By the ninth day patient had nearly recovered all her power and sensation in right leg and arm. Some facial paralysis still remained, the mouth being drawn to the left, and the tongue when protruded, directed to the right. January 15, patient had very offensive expectoration. Coarse and fine râles over lungs. January 17, expectoration very offensive. Slight dullness and broncho-vesicular breathing, with slightly exaggerated voice sounds over part of right lobe, posteriorly. January 19, marked dullness and marked bronchial and bronchophony over upper part of right lower lobe. Expectoration so offensive that patient was removed to a retired corner of the ward. Temperature 101° to 102°.

Patient grew weaker every day. She died on the 21st, thirty-seven days after admission.

Autopsy. Brain.—Slight atheroma of arteries at base. Left corpus striatum contains a small reddish, fibrinous clot within a sac, and around this the brain tissue was indurated to a slight degree and of a yellowish-red color. The position of the clot was in lenticular nucleus of corpus striatum. It was of oblong shape, and slightly involved adjacent island of Reil and the tissue beneath the nucleus. The white matter of the capsule had not been lacerated by clot, but simply affected by pressure.

Lungs.—Right lung was consolidated throughout, of gray hepatization. Two spots of gangrene in upper and one in lower lobe. The dead tissue was not entirely separated, of a grayish-white color. All the spots were round, or wedge-shaped, and touched pleura. The pleura on this side contained about ℥ lxxx of serum which was mostly between upper lobe anteriorly and chest wall, the adhesions below preventing the accumulation below.

Heart.—Normal. *Liver.*—Slightly fatty, depressed and marked by a slight sac depression, producing an elongation of right lobe reaching nearly to crest of ilium. *Spleen.*—Small. *Kidneys.*—Nothing marked.

Case II. Male, 59, left hemiplegia with exaggerated reflexes. Gradual mental failure, death on twenty-seventh day. Lesion of right lenticular nucleus, most marked in putamen, but involving globus pallidus and internal capsule somewhat. Temperature normal.

Patient was admitted on October 23.

Family History.—Father was insane; otherwise negative.

Previous History.—Had syphilis twenty-four years ago, and had been a heavy drinker of brandy and whiskey for many years. Five years ago had rheumatism. Has otherwise been well except for present trouble.

Present Illness.—October 22, the patient felt strong and well when he retired, but in the morning when he arose, he felt a peculiar numbness in the left side, and on attempting to stand, he fell over on account of loss of power in left side. Says he has been unable to use the left arm or leg since and that his head feels peculiar. Thinks he can not speak as readily as before the attack, but can say what he wishes, and has no loss of memory of words. Feels more drowsy than he did before the attack.

Physical Examination.—Patient was well nourished, average size, not anemic, tongue moist, slightly coated. In both hands there was a fine tremor and some ataxia, the ataxia being the more marked on the left. There was marked loss of power in the flexor and extensor muscles of the left hand. The plantar reflexes were exaggerated, and there was marked ankle-colonus on the left side. The left patellar reflex was exaggerated; the right one normal. The skin reflexes were absent on the left side, except epigastric reflex which was diminished. The patient could not stand alone, but there was some strength in both the legs.

Urine, acid, 1024, amber, no albumin, leucocytes, uric acid, polygonal cells, sed. mucus.

November 15, patient has been gradually failing in strength since admission, had incontinence of urine and feces. November 18, patient died twenty-seven days after attack.

Autopsy.—November 21, rigor mortis fairly well marked. The dura mater was quite adherent to the cranial bones, especially at the posterior portion. The arachnoid was much thickened, and of a whitish color. The vessels at the base were atheromatous. The brain was then cut in transverse sections according to Pitres' method. In the outer segment of the right lenticular nucleus was a patch of softening, about the size of a bean. There were also many capillary extravasations throughout this nucleus and in the internal capsule. The left brain was normal. Both lungs were somewhat congested, the right being bound down by slight adhesions at both apex and base; otherwise they were normal. The heart was somewhat flabby and slightly fatty; otherwise normal. The liver was very firm, of about normal size, and had some excess of interstitial tissue. Spleen somewhat soft, but of normal size. Kidneys were somewhat diminished in size, capsules slightly adherent, markings indistinct, others dusky and of irregular outline. No fluid was found in any of the cavities, save the pericardial, and this could not be considered in excess of the normal.

Case III. Male, aged 43. Left hemiplegia continuing for a year, but permitting patient to work. Eight months later right hemiplegia, which was temporary, patient returning to work. Finally ventricular hemorrhage and death on same day. Cysts found in both lenticular nuclei, larger in right.

Family and early personal history not obtainable. About one year ago the patient had an attack of apoplexy, which left him with right hemiplegia for some time. He partially recovered from this, but had some loss of power persisting. He returned to work, but was obliged to accustom himself to work with the left hand. Four months ago he had another attack which resulted in paresis of the left half of the body. He entirely recovered the use of this side. For some weeks patient has had indefinite sensations of illness, such as formication paresthesia in different parts of the body, and has suffered from headache, but he was able to continue his work. On the day of admission he was at work in the morning, but complained of weakness and mental confusion. He went into a beer saloon and drank one glass of beer; he remained there intending to rest himself. He was then seen suddenly to fall from his chair and become unconscious. He was immediately sent to the Reception Hospital, at 99th Street, and from there transferred to Bellevue.

Examination on Admission.—Patient was unconscious, somewhat delirious and restless. Pupils contracted irregularly, the right less than the left. Loss of power is noted in the left lower extremity; the other limbs are somewhat rigid, except when voluntarily moved by patient. Patient rolls the eyes upward and to the right, and the head is turned to the right, with rigidity of the cervical muscles. Urine is passed involuntarily, and the respirations are slow, with inclination to stertor. Temperature 102°; pulse full and about 85. Patient remained in the cells until the following morning, when he developed pulmonary edema, and was transferred to the hospital wards. A small quantity of urine was obtained by use of catheter, and was found to be loaded with albumin. The patient died on the night of admission.

Autopsy.—Dura mater thickened and adherent. Pia mater opaque in many places, and beneath it a large quantity of blood was effused, distributed over entire surface of brain, but chiefly seen at the base.

Brain.—An extravasation of blood was found in the lenticular optic region of the right side. The tubercula quadrigemina were somewhat torn, especially on the right side. The ventricles, lateral, 3d and 4th, were filled with clotted blood, and blood had escaped from these beneath the pia mater over the brain surface. The internal capsule was little injured, being slightly torn posteriorly. In the lenticular nucleus of each side were cysts, the remains of former apoplexies—that on the right side being much larger than that on the left.

Heart.—Left ventricle hypertrophied largely. A small calcareous spot existed at base of mitral valve.

Lungs.—Congested and edematous.

Liver.—Cirrhotic and fatty.

Kidneys.—Presented interstitial and fatty changes. Capsule separated with difficulty, leaving surface torn and granulated.

CASE IV. Male, aged 73, admitted in a condition of semicoma, no distinct signs of paralysis, but a general rigidity and later convulsive movements, relieved by bleeding. Death on same day. Bilateral softening of both lenticular nuclei, especially involving the globi pallidi.

Patient was admitted to the hospital March 9. No history. He could be partly roused to a consciousness of his surroundings, but was unable to respond to questions. Face was flushed; breathing quiet. Head and eyes turned to the right. Pupils moderately dilated and even. Marked rigidity of all the limbs, most pronounced in the left arm. The left hand and fingers are held rigid, the fingers being flexed at the first phalanx and the second and third extended. There was no distinct evidence of paralysis. He did not respond to cutaneous irritation, probably on account of the stupor. He was able to swallow. The heart action was weak; the urine showed specific gravity 1026, albumin 0.1 per cent., no casts noted, but phosphates and a few pus cells. Spleen and liver normal. Twenty-four hours after admission the patient had slight spasms, beginning with convulsive movements of the face and left hand, and including movements of face, head and both arms and slighter movements of the legs. They were considered uremic, though there was little albumin and no casts or edema. He was bled 7½ ounces, and immediately after the patient seemed more conscious. The pulse continued weak. The spasms ceased. The record shows that after this the stupor gradually deepened again, and the patient became comatose, dying early the third day. The temperature record of the three days that he was in the hospital was taken in each axilla and in the rectum. The temperature was 101.2 the first day 100 to 99.8 on the second, 102 on the third.

Autopsy.—The full record of the autopsy was lost, but the notes made by myself record two spots of softenings, one in each lenticular nucleus, and especially involving the globus pallidus.

The Effects of Gas Poisoning on the Lenticular Nuclei.—It has long been known that a very common, if not almost universal, effect of sewer gas poisoning, is a softening of both lenticular nuclei. This may happen if the patient has been unconscious from the effects of gas for more than twenty-four hours.

It has seemed to me that a profitable method of studying the functions of the lenticular nuclei might be that of observing the physical and mental condition of patients who have suffered from severe gas poisoning.

Ziemssen first reported a case of gas poisoning with softening of the corpus striatum in his "Electricity in Medicine," in 1864.

In 1868, Th. Simon (15) showed that gas poisoning caused thromboses and softening of the brain and particularly of the lenticular optic region. He describes first, I think, bilateral lesions of the lenticular nucleus as a result of this condition.

A woman, aged 46, was found unconscious and asphyxiated from CO_2. The poisoning was very severe and consciousness was not restored for several days. She finally recovered and resumed her work for a few days, then developed symptoms of mental disturbance. She lay with limbs stiffly outstretched, groaning and screaming, and had some convulsive attacks. Pupil, left eye, contracted to point, right eye gone. Pulse 88. Answered no questions, swallowed little, spit out food put in her mouth. She died in seventeen hours. Autopsy showed in the left side of the brain a spot of softening the size of a cherry in the middle of the corpus striatum which at the limit of the thalamus behind was more solid. A similar, but small lesion was in the right striatum. No other spots of softening.

In 1898, Kolisko published a series of articles upon the blood vessels of the brain. In this he particularly called attention to the existence of a special artery which is given off from the anterior cerebral, near the communicating branch. This artery is usually single, but sometimes double. It runs upwards and backwards, and it supplies the caudate nucleus, part of the anterior portion of the internal capsule, and part of the outer portion of the lenticular nucleus. Owing to the length and peculiar direction of this artery, the blood that passes into it has to flow rather against the normal current. The result is that the pressure in this artery easily becomes relatively less than that of the other vessels, and when the general blood pressure is very low, as in gas poisoning, a tendency to stasis and thrombosis occurs. This artery is the one, then, that is particularly selected as that in which thromboses occur, and Kolisko states that it has as much right to the name of "the artery of cerebral thrombosis" as the branch from the middle cerebral has to the name of the "artery of cerebral hemorrhage." It is an involvement of this artery on each side, which occurs in cases of gas poisoning, and which leads to the very common lesion of bilateral softening of the corpora striata in that condition.

The Syndrome of Lenticular Lesion.—Klebs (16) quotes a case. Man poisoned by gas and under its influence four days. Had paralysis of the right arm, edema and gangrene of skin. He had a sharply limited focus of softening in the corpus striatum. He also reports two cases of symmetrical softening of the caudate nuclei and the two inner parts of the lenticular nucleus and anterior part of the internal capsule in which vaso-motor and trophic disturbances of the skin occurred.

Kahler (17) reports a similar case and the findings are shown in Lesser's Atlas, II, Tafel xviii, case 7.

Poelchen and Kolisko (18) observed the same phenomenon.

The study of the cases reported shows that with the involvement of this region, there often occur edema and gangrene of the skin (Klebs, Kahler, Porechen, Kolisko). So that when after gas poisoning one finds the patient unconscious with these vaso-motor and trophic disturbances present, a diagnosis of lenticular lesion may be made—Kolisko did actually make such a diagnosis, verified by autopsy.

It may therefore perhaps be said that severe symmetrical softening of the corpus striatum has a " syndrome." If a person poisoned by gas is made unconscious for over forty hours and develops skin lesions, vaso-motorial and gangrenous, he has a double lesion of the corpus striatum.

Clinical Study of Recovered Cases.—A further and special object which I had in view in studying this subject was to find out the later history of severe gas-poisoning in which recovery took place. It may be that in these patients the lenticular nuclei have been injured and thus a pathological experiment is made. I have tried to get hold of all the recovered cases that had been brought into Bellevue Hospital in the past year, but I was not very successful, as only four patients were examined. Besides these I had under observation several years ago a victim of gas. In this case there was a prolonged period of amnesia, with the development of practically a double personality. The case was reported by me at length in the *Psychological Review.*

Another patient was well except for curious attacks of vomiting which would come on explosively, without any particular cause. The other two said that they were as well as they had ever been in their lives. There is in the literature of gas poisoning only some references to the fact that it leads to impairment of memory. My investigations so far have not been very fruitful, but it seems to me that a further examination of these cases by neurologists may lead to some interesting results.

It is well known, of course, that other and severe lesions of the brain may be caused by gas poisoning, but the most common and practically uniform lesion is that of the lenticular nucleus. This lesion need not be a very large one and in fact, one must examine closely sometimes to find it. I have photographs of a brain with lenticular softenings from gas poisoning furnished me by the kindness of Dr. Otto Schultz and Dr. James Ewing, upon whose authority I make my statements regarding this condition.

Summary.—The corpus striatum has not any independent or specific motor function. It probably has some supplementary motor function, and especially in connection with articulation.

It may have some control over the bladder (double lesions).

It seems to have some control over vaso-motor and trophic conditions of the skin (and lungs?).

It has no thermic center.

It may have some supplementary and associative psychic function so that lesions affect memory or initiative.

It is an organ of less importance relatively in the higher vertebrates.

In severe gas poisoning there is a double softening of the lenticular nuclei due to thrombosis of "the artery of cerebral thrombosis," and there result vaso-motor and gangrenous conditions of the skin, so that these conditions in connection with a history of coma from gas poisoning form a group of symptoms called "the syndrome of the corpus striatum."

1. Sellier and Verger. Rev. Neurologique, 1903, p. 736.
2. Schüller. Jahrbüch f. Psychiat. and Neur., Vol. 22, p. 90, 1902.
3. Tounel and Raviart. Echo Med. de Nord, p. 137, 1897.
4. Stida. Rev. (russe) de Psychiatrics, de Neur. and de Psychol. Exp., 1902, No. 8, and Neurol. Centralb., No. 8, April 16, 1903.
5. Chailchevitch. Ibid., No. 9, —, 686, 1902.
6. Lo Monaco and Bellanova. Riforma Medica, 8–9, October, 1902.
7. Mingazzini. Rivist. Speriment di Frenat., 1902, xxvii, fasc. 2, p. 484; also p. 317.
8. Löwy. Deutsche mediz. Zeit., Nos. 71–72, 1903.
9. Touche. Arch. de Neurol., XIII, p. 467, 1902.
10. Marburg. Neurol. Centralb., 1904, p. 781.
11. Czharlz. Wien klin. Woch., 1902, No. 31, p. 788.
12. V. Bechterew. Neur. Centralb., 1905, No. 17, 786.
13. Franceschi. Rivista di Path. Nerv. e Ment., 1905, p. 249.
14. Pagano. Rivista di Path. Nerv. e Ment., Vol. XI, fasc. 7, p. 289, 1906.
15. Simon. Arch. f. Psych., Vol. I, p. 263.
16. Klebs. Virchow's Archiv., xxxii, 502.
17. Kahler. Prag. med. Woch., 1881, No. 48.
18. Poelchen and Kolisko. Hoffmann's Lehrbuch, p. 739.